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ABSTRACT

The diagnostic problem presented by children without obvious neurological, cognitive, genetic, emotional or environmental basis for their atypical or delayed language development is discussed. One unresolved issue is whether the deficits of such dysphasic children are linguistic or are more fundamental cognitive or perceptuomotor deficits. A second important issue is whether the differences in language capabilities between normal and language-disordered children are qualitative differences or quantitative ones. It is suggested here that language disabilities derive from genetic idiosyncracies leading to neural systems with differential capabilities for acquiring language. Children with deficits in language, because of their idiosyncratic makeup, may have to struggle to learn the acquisition strategies innate for the majority. It is suggested that the needs of the speech clinician might better be met by recognizing that dysphasic children possess different patterns of neural organization that predispose them to approach language learning in an atypical fashion. (CLK)

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Idiosyncratic Genetic Specificity for Neurolinguistic Systems:
A Cause of Atypical or Delayed Language Acquisition

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A vexing diagnostic problem is presented by those many children exhibiting delayed or atypical language development without any obvious neurological, cognitive, genetic, emotional, or environmental basis. Such children often exhibit the same types of deviant language behavior found with "secondary speech disorders" resulting from mental retardation, Down's syndrome, infantile autism, childhood psychosis, or childhood aphasia. In addition to atypical development of language, many of these children also manifest deviant perceptuo-motor processing, short-term memory disabilities, as well as deficits in general and particular aspects of cognitive development.

A major unresolved issue is whether the disorders these dysphasic children have are in essence linguistic deficits versus either a more fundamental cognitive or perceptuomotor deficit. Also a matter of dispute is whether the differences between the language capabilities of normal and language disordered children are qualitative or quantitative: evidence has been marshalled on both sides of the argument. To put both of these controversies in perspective, it is perhaps useful to remember that any attempt to provide a single diagnostic or etiological answer for this entire class of children is doomed to failure. There is reason to expect that virtually any combination of quantitative and qualitative differences in linguistic and nonlinguistic processing could be identified in one subset of children or another.

Perhaps the most frequently invoked diagnosis for children with specific language disabilities has been brain damage. This diagnosis is often made even though these children (by definition) exhibit none of the major neurological signs

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of pathologic brain malfunction. There has been a heavy reliance on certain "soft" neurological signs as indicating the existence of subtle, minimal brain damage in dysphasic children. Many clinicians have argued convincingly that the use of soft signs in diagnosing minimal brain damage is questionable and there remains no legitimate evidence that brain damage is responsible for the language problems of any significant percentage of these children. Given all that is known about the effects of damage to immature neural systems, it is highly improbable that subtle injury to preoperational neurolinguistic or cognitive systems before or around the time of birth would later produce deviant patterns of language development.

A second approach to diagnosing the problems of this behaviorally defined class of children has been to claim that it is abnormal functional activity that is responsible for atypical language acquisition. One view sees the problem as being one of cerebral dominance: either mixed dominance, delayed dominance, or failure to establish full laterality. Another view suggests that these children have chosen the visual modality as the preferred learning modality, with resulting difficulties for processing auditorially encoded speech. Both views fail to account for the normal language development of many other children with these same functional specializations.

A large number of language disordered children exhibit functional deficits in high-level auditory processing of temporal sequences and/or deficits in short-term memory processing. Both of these functional domains have been put forth as the primary deficit of language disordered children. Any such proposal is limited in its diagnostic power by the failure to eliminate the possibility that human beings possess high-level auditory processing capabilities and complex short-term memory strategies for verbal input only as the RESULT of language capabilities and not as the necessary basis for the acquisition of language.

Perhaps it is because language is deficient in these children that they fail to develop sophisticated auditory or short-term memory strategies. Before any definite conclusion can be reached, some means must be found to resolve this chicken and egg question.

The attempt to show that these children do not have a linguistic deficit as their primary problem reflects in part a backlash against the recently resurrected view that much of language structure is innate. However, there is much evidence showing that language and speech are in fact part of the biologically determined maturational schedule of our species. This is true whatever the (dubious) merits of Chomsky's claims concerning the competence/performance distinction and substantive and formal universals as constituting an innate Language Acquisition Device. There can be no doubt that genetically specified neural systems with language functions exist and lead to a regular progression of developmental stages of language acquisition in children from widely varying cultures and environmental circumstances. Dysphasic children are identified as such precisely because they deviate from species-wide norms. Whether language development is based on specifically linguistic neural systems or more general cognitive systems, it is clear that these systems are biologically based in Homo sapiens. Since there is no time here to fully justify a neurobiologically formulated nativist position, let me state it in the form of a hypothesis basic to the conclusions of this paper. This statement is given as Number (1) on the handout.

There is no requirement that innate neurolinguistic systems be present at birth, but only that there be encoded in the genetic material information that triggers the development and regulates the operation of these systems at appropriate stages of maturation in particular sorts of environments. As one examines the maturation of the human infant, it can be observed that some

neural systems require little interaction with the external environment (that is, they are highly specified in the genotype), while others require a greater degree of interaction with the environment and are subject to greater variability of mature form (that is, they are less highly specified genetically). Since all phenotypic traits require both a genetically specified basis as well as some sort of interaction with the environment in which development takes place, the difference between innate traits and learned traits can only be a matter of degree. This fact is summarized in number (2) on the handout.

With the exception of identical twins, we are all genetically unique. Those traits that psycholinguists present as "universals of child language acquisition" are in actuality only approximations of the typical behavior for members of the species as a whole. Additionally, universality is not a necessary grounds for inferring that a given behavioral trait has a highly specified genetic basis. It is entirely possible that a given trait could develop from an innate genetic basis in one segment of the population and from a greater degree of individual learning in some other segment. Both groups would exhibit approximately the same mature behavior, even though they achieved it in different ways. While it is simpler to talk about how "the child" acquires language, it is more realistic to begin with the assumption that qualitative and quantitative differences in neurolinguistic systems will produce a wide range of individual differences with many means for accomplishing the same goal. This view is summarized in number (3) on the handout.

The innateness question can be answered only for particular domains of neural information processing and only on an individual basis. One implication of this fact is that it is a mistake to presume that the deviance from species' norms in dysphasic children is necessarily a reflection of "abnormal" or

"disordered" neurolinguistic or cognitive systems, when all that may be at issue is that their development is "atypical." An obvious, but neglected explanation for the deviant language development in a large number of dysphasic children is that, rather than being the product of neural disorders, language disabilities may actually derive from genetic idiosyncracies leading to neural systems with differential capabilities and strategies for acquiring language. The reasonableness of the view that the phenotypic capabilities of neurolinguistic systems derives from idiosyncratic genetic specification of these systems depends on the degree to which language capabilities can be associated with genetic factors.

It has been known for a long time that language problems tend to run in families and that global difficulties in acquiring language can be attributed to inherited characteristics. As discussed in Lenneberg (1967), the familial occurrence of congenital language disabilities is well-documented in a number of published pedigrees. It seems clear that developmental dyslexia can be related to genetic factors and that delay in the onset of speech is much more prevalent in some families than in the population at large. Luchsinger (1959) established the familial occurrence of word deafness, speech sound deafness, and congenital difficulties with the acquisition of syntax. Especially revealing are the many twin studies showing that fraternal twins are much more likely to exhibit differences in language acquisition patterns than identical twins. Identical twins exhibit similarity in the time of speech onset and in speech milestones 90% of the time as against only 40% of the time for fraternal twins. Identical twins show the same delay in speech onset 65% of the time versus only 35% for fraternal twins. One may reasonably agree with Eustis (1947) that genetic factors provide the primary etiology of congenital language disabilities, but the basis for the inheritability of language deficits or

delayed language development need not be chromosomal abnormalities, nor any sort of disorder at all. As summarized in number (4) on the handout, the genetic variability found in our species is in itself sufficient to explain the existence of varying capacities to acquire language. While it is still necessary to diagnose the functional deficit a given child has relative to the species' norm, one need look no further to find the etiological basis for a great many children's behavioral disabilities in language.

I would like to move this explanation one step further back in the causal chain and show why this situation is to be expected, and in fact necessary for the well-being of our species. My proposal draws on the fact that any genetically specified neurofunctional systems were, at one time in the species' history, not genetically specified. Innate neurolinguistic systems have their roots in the evolution of Homo sapiens and the capacity of our species for language, as a reflection of the social mode of adaptation of our hominid ancestors and the complex symbolic communication systems that were necessary to support a complex social organization.

One realm of functional specialization of the mammalian nervous system is that of communication between members of social species. All mammals homologously share certain patterns of communicative behavior based on the activity of the limbic system, a forebrain complex regulating our appetites, emotions, arousal, motivation, and social interactions. The communication behavior of the human infant during the first postnatal year is not substantially different from that of other primates, and the capacity of the human child to advance beyond this limbic level and acquire a language system is the result of evolutionary changes in neocortical organization that have occurred during the past 20 million years or so of separate hominid evolution.

Linguistic communication systems and the neurofunctional systems that allow them to be acquired and used by human beings did not evolve all at once. Both arose in an integrated series of mutually supportive stages in the evolution of culture, cognition, communication, and the brain. While there is little direct evidence on how the evolution of language (and the brain systems that support it) took place, it seems an inescapable conclusion that at any stage of hominid evolution, our ancestors exhibited a wide range of genetic variability. We may also expect that this variability was manifested in the nature of the neocortical systems utilized to acquire non-limbic verbal communication systems. Thus, it is highly probable that some infants were able to acquire the communication system of their culture more easily than other infants, based on the possession of neurofunctional systems adapted to carry out this sort of information processing activity.

At all stages of hominid evolution, a necessary prerequisite was that infants have the ability to learn the communication system of their environment easily and without formal training. A survival oriented, subsistence level hominid culture could not afford the social resources to send children to school for years just to learn to communicate. Either a given infant picked up the system through exposure, or it would be ill-equipped to function as a member of the group. Our survival as individuals depends on our place in a network of social relationships. For this reason, there is an adaptive value in being able to communicate efficiently with other members of the species. This could have been no less true for our hominid ancestors. Those individuals with the genetically based neural idiosyncracies that are up to the task of acquiring the communication system of the environment in an efficient manner would have had a natural selective advantage precisely to the degree that it was maladaptive to be unable to learn to communicate efficiently. Individuals with adaptive

idiosyncracies would tend to propagate their kind at the expense of those who, due to their inability to communicate, could not participate fully in cultural life. Eventually this would lead to a concentration in the species' gene pool of the adaptive genetic idiosyncracies. The result over time would have been an increasingly greater percentage of infants who shared adaptive neocortical communication capabilities. In terms of the ontogenetic schedule, such a process would likely come to be manifested as a tacking on of a new stage in the maturation of the neural systems making up the communication hierarchy. At some point in ontogenetic development, these neural systems would become operational and allow the acquisition of the communication system of the environment.

When the majority of the members of the species had this same advantage and learned to communicate quickly and easily, then and only then would the basis have existed for the cultural invention of a new, higher order communication system. Once again some individuals would have had the advantage of being able to learn this new system easily through mere exposure. This would have led to a repetition of the cycle and eventually allowed descendants many times removed to acquire linguistic systems with syntactic and morphological structures. The means for the inheritability of this capacity would have been the incorporation into the genetic material of reflexes of the adaptive neural idiosyncracies of our ancestors. This scenario is summarized in number (5) on page one and on page three of the handout.

The result of such a process over 20 million years would be a maturational schedule for descendants that included a whole series of prematurational stages in the development of the verbal and nonverbal communication systems that characterize our species. Page two of the handout summarizes the schedule of stages of communication development for the average member of our modern species.

Each phylogenetic stage in the evolution of adaptive neural systems in immature ancestors finds its reflex in the maturational schedule in a modified recapitulative fashion. Among other things, this scenario explains why we find in modern Homo sapiens a language acquisition process that includes a series of prelanguage stages that clearly derive intrinsically from the infant and not from the environment.

Of course it is a cruel misfortune that some children develop language disabilities due to their genetic makeup, but from the viewpoint of the species as a whole, genetic variability is necessary in order to retain the potential for acquiring new capabilities and adapting to novel environmental circumstances. Adaptation through cultural and social means based on individual learning can go just so far. At some point there must be actual organizational changes in neural equipment for the entire species to allow new high level capacities to arise. These organizational changes require as their basis the prior existence of individuals possessing these specialized neural patterns as their idiosyncratic legacy.

The reflexes of older evolutionary stages are 'buried' lower down both in the genetic material and in the behavioral progression of developmental stages. Each species-wide developmental stage represents a commitment that the individual cannot escape from. Because they have been subject to selective pressures for millions of years, lower level systems can be expected to exhibit less variation between individuals as compared with higher level, more recently evolved systems. For the progression of stages in the acquisition of verbal communication systems, this means that it is particularly in the acquisition of syntactic and morphological structures that the highest degree of variability would be manifested. Even severely retarded children go through fairly normal holophrastic, two-word, and telegraphic stages (however delayed the onset or extended a given stage might be).

Individual variability would also be likely to be found in the higher level auditory and short-term memory processing involved in speech comprehension, as well as in the complex neuromotor systems responsible for speech production. We would expect the range of individual differences to be particularly wide for reading and writing. These skills have not existed long enough for our species to have evolved specialized neurofunctional systems precisely to carry them out.

In summary, the reason why many children have deficits in language may simply be that, because of their idiosyncratic makeup, they do not receive the same degree or type of specialized help in acquiring language as the average child. These children must struggle to learn the acquisition strategies that are innate for the majority. Rather than inventing labels for nonexistent disorders, the needs of the speech clinician might better be met by recognizing that these children merely possess different patterns of neural organization that predispose them to approach language learning in an atypical fashion.

HANDOUT FOR

Idiosyncratic Genetic Specificity for Neurolinguistic Systems:
A Cause of Atypical or Delayed Language Acquisition

SUMMARY STATEMENTS:

- (1) INNATENESS As a species-specific characteristic, there exist innate neurofunctional systems that provide the necessary foundation for the acquisition of language by children. These neurolinguistic systems organize in human ontogeny according to a genetically regulated maturational schedule of stages, providing that appropriate environmental experience occurs.
- (2) DEGREES OF GENETIC SPECIFICATION A given neurofunctional system is innate to the degree that there is specific encoding of information in the genotype that causes the differentiation and organization of that system during ontogenetic maturation. The level of environmental experience necessary and the potential for variable results in the nature of the developing system is a measure of the degree of genetic specificity (or innateness) of that system.
- (3) IDIOSYNCRATIC GENETIC SPECIFICITY All members of our species can be expected to exhibit idiosyncratic specification for genetic triggers of neural maturation. Neurolinguistic systems are encoded in the genetic material of different individuals to varying degrees and in diverse manners. While we may observationally identify species' averages for the time at which a given language system becomes operational, or for the span of its maturation, all children may be viewed as distributed along some developmental curve that represents the various phenotypic realizations of idiosyncratically specified neurolinguistic systems.
- (4) IDIOSYNCRATIC GENETIC SPECIFICITY AS A CAUSE OF DEVIANT LANGUAGE DEVELOPMENT Relative to the species' average, we may classify certain children as having a language disability that may be pervasive or restricted to one or another domain of language processing. The primary cause of atypical or delayed language acquisition in a major segment of such children may be the idiosyncratic genetic specificity of their neurolinguistic systems. Because they possess different innate neurolinguistic systems, some children acquire language more quickly or more slowly than the species' norm, and in a particular domain of language processing a given child may possess strategies that are more or less efficient than the species' norm. A predisposition toward a particular pattern of language acquisition can be inherited and there is no need to posit either neural damage, functional disorders, or chromosomal abnormalities to explain the range of differences observable in many children with atypical language development.
- (5) ADAPTIVE NEURAL IDIOSYNCRACIES AS THE BASIS FOR LANGUAGE EVOLUTION Given the importance of communication for ancestral hominids with a social mode of adaptation, there would have been a tendency for the concentration in the species' gene pool of those genetic idiosyncracies that promoted the efficient acquisition of the culture's communication system by infants. Such a process would lead to the sharing of species-wide traits of neurofunctional organization and thus provide the basis for the cultural invention of new, higher order communication systems. Repetition of this cycle led eventually to language as we know it. Adaptive neurofunctional systems would become incorporated into the genetic material of descendants and thereafter be transmitted by heredity as stages in the maturation of the descendant's communicative competence.

(from Lamendella, 1975)

OUTLINE OF THE
MATURATIONAL STAGES IN THE DEVELOPMENT OF
COMMUNICATION SYSTEMS BY THE CHILD

I. PRECONCEPTUAL LIMBIC STAGES

From about two weeks postnatally, the infant exhibits a fixed set of multimodal sign complexes that allow a receiver to infer a closed set of graded messages. The perception of specified internal or external conditions results in the automatic implementation of a set of differentiated responses produced by action schemata of subcortical components of the limbic system. From about 6 mo., higher level paleocortical limbic components integrate with the neocortical motor systems that control voluntary motor activity to allow the intentional production of existing limbic responses as a conscious means of communicating motivational states to those in the environment. The infant becomes more responsive to the affective and conative content of adult behavior and, from about 9 mo., prosodic features such as intonation contours become part of limbic signal complexes. Certain universal limbic 'words' (e.g., man "food"), while retaining roughly the same phonetic form, make the transition from involuntary sign to intentional signal.

II. PRELANGUAGE COMMUNICATION SYSTEMS

A. Gestural Stages: Multimodal gesture complexes output by secondary neocortical movement schemata are systematically and willfully used in particular contexts to communicate the substantive and relational content of an open set of propositional conceptualizations. As various components of gesture complexes become non-representational and conventionalized, the transition is made from signal to symbol. Vocalizations have no special high status as against the other components of a given schema such as facial expression, visual orientation, body orientation, body configuration, rhythmic and patterned movements.

B. Phonological Labeling Stages (Naming Stage): Phonologically structured names act as labels on object-level and propositional concepts in such a way that hearing a given phonological form can lead to the retrieval of a concept from long-term memory. Perceptions that lead to the conceptual recognition or recall of a given generic or token concept may result in the overt vocal production of the child's label for that concept. Such articulatory output is organized by lateralized neocortical movement schemata based in Broca's convolution. Though apparently not used to communicate propositional messages, labeling vocalizations are used in social interactions to identify objects and as vocatives.

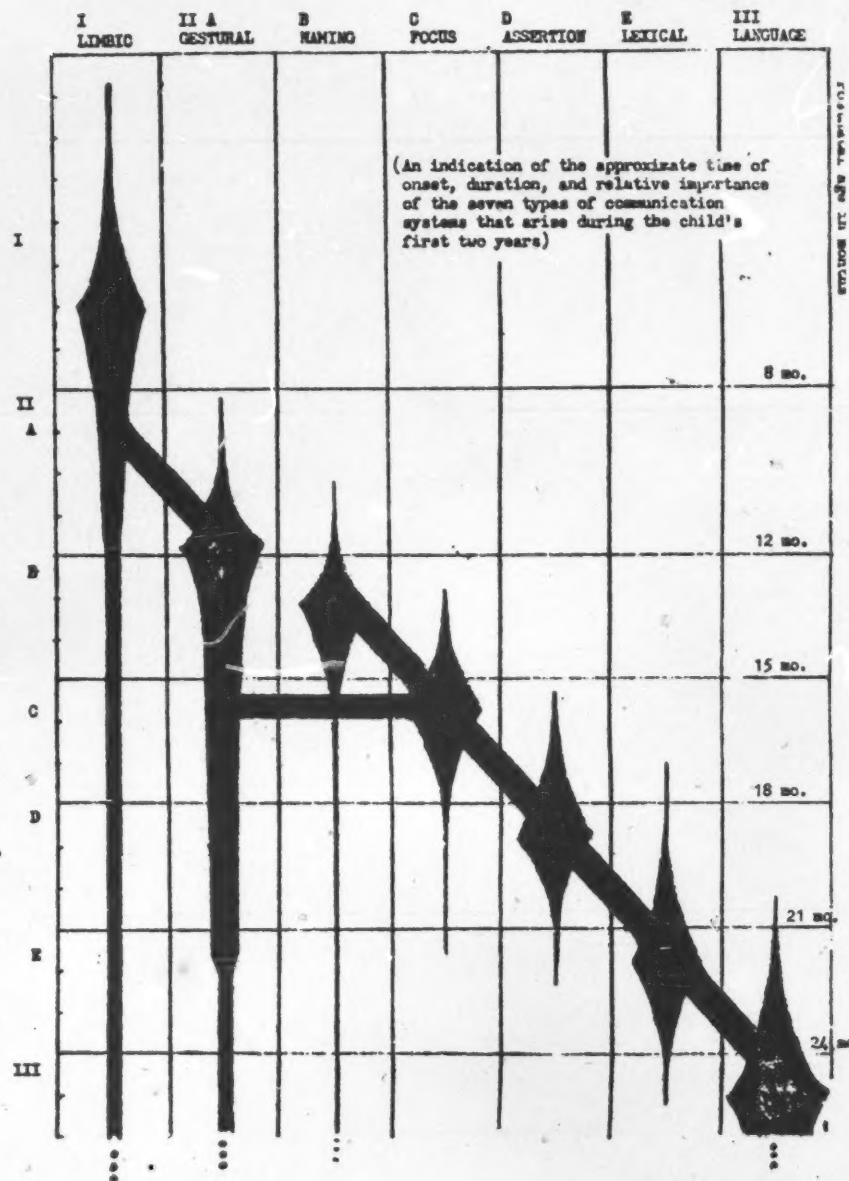
C. Propositional Focus Stages (Holophrastic Stage): Neocortically based communication systems insert phonologically structured labels into previously existing gesture complexes. One function of the verbal component of a gesture complex is to draw an addressee's attention to the information focus of a given propositional message. The conceptual content of one word utterances becomes increasingly differentiated over and above that of the gestural substratum. For the most part, both the messages the child encodes and those decoded from adult speech are still approached in terms of an immediate action strategy.

D. Propositional Focus-Assertion Stages (Pivot-Open Stage, Two-Word Stage) Two word utterances accompany gestural complexes, functioning to identify the information focus and also to make an assertion or predication regarding this focus. While relational concepts are still expressed almost exclusively by gestures, or by relying on the contextual situation, the child increasingly opts for the auditory-vocal communication channel. Vocalizations more and more begin to independently communicate substantive concepts that are part of the message.

E. Lexical Stages (Telegraphic Stage): The mean length of utterances goes up as the child outputs strings of lexical items capable of assuming a major burden in communicating the substantive content of messages including some relational notions. No syntactic structure exists, although word order may be fixed. Even though lexical strings remain subject to misinterpretation, the limbic and gestural systems are of diminished importance in the child's overall communicative repertoire.

III. LINGUISTIC STAGES

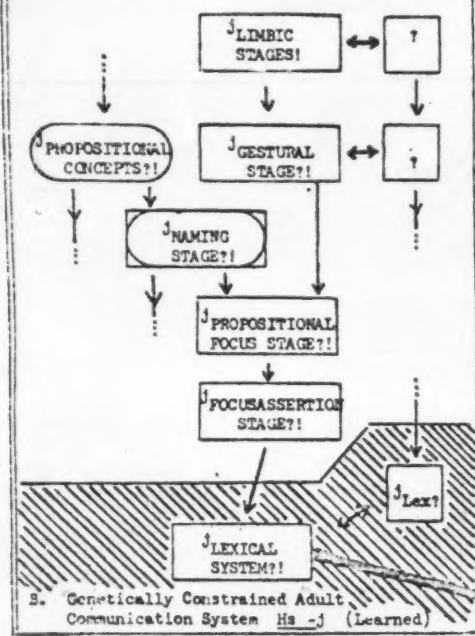
Lateralized neocortical systems produce sentences that communicate both relations and substantives by means of syntactic devices and grammatical morphemes used with lexical items. In general, the conceptual grasp of a given distinction precedes its linguistic expression. The mastery of linguistic forms is subject to certain universal constraints. Sentences become increasingly independent of accompanying gestures and situational context.



Hominid Precursor Hs -j

A. Genetically Regulated Prematurational Stages for Hs -j (Innate)

COGNITION COMMUNICATION
Production Comprehension



Credibility Key

- . = observable
- ! = empirically based claim
- !? = probable inference
- ?! = plausible inference
- ? = speculation

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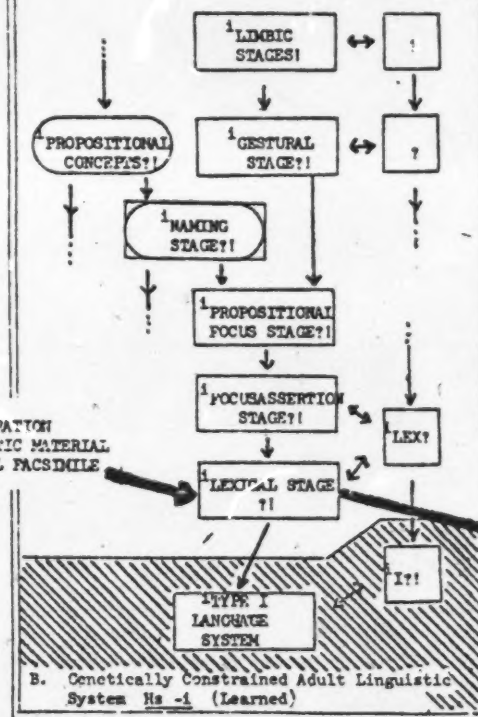
The example above posits two specific hominid ancestors in order to illustrate the general claim that as more complex symbolic communication systems became adaptive for hominid subspecies, selection for appropriate sorts of genotypes led to the additive incorporation of new prematurational stages in the acquisition of communication systems into the ontogenetic development of descendants. This in turn provided the basis for the phenotypic learning of a qualitatively new order of communication system and, in cyclic fashion, to the eventual incorporation of a reflex of this system into the genetic material of the evolving species. For the descendants, the new stage in the acquisition of the adult communication system would, in all likelihood, not be identical to the ancestral system, but would be some homologous diachronic transformation of the system it recapitulates. Needless to say, each specific claim for the status of a communication stage in the modern child as a vestigial repetition or vestigial facsimile is subject to independent empirical verification.

Even in the absence of any applicable comparative data from other modern primates, or direct neurological evidence from fossil remains, it would be possible to formulate probabilistic reconstructions of stages in the phylogenetic evolution of human linguistic communication systems by projecting backwards from the innate, universal stages observable in the acquisition of language by children. It is hypothesized that some of these stages exist only as modified repetitions of the prematurational ontogenetic stages in the development of communication systems by some hominid precursors. (from Lamendella, in press-a)

Hominid Precursor Hs -i

A. Genetically Regulated Prematurational Stages for Hs -i (Innate)

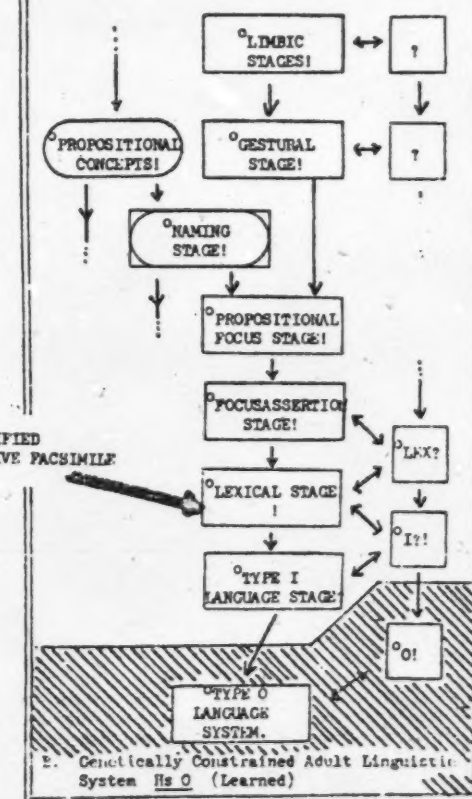
COGNITION COMMUNICATION
Production Comprehension



Homo sapiens sapiens Hs 0

A. Genetically Regulated Prematurational Stages for Hs 0 (Innate)

COGNITION COMMUNICATION
Production Comprehension



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